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A Modelling of Emerging and Re-Emerging Infectious Diseases

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Abstract: ***In the field of epidemiology, the Susceptible, Exposed, Infected (SEI) model was developed with the aim of understanding the type of diseases that have a character of the individual infected, remains and remains contagious until the end of his/her life. But the phenomenon generated by global warming has changed the behavior of our biodiversity and ecology of the transmission of infectious agents. This causes the adaptation of the current existing model facing new behavior of infectious diseases. In this paper, we propose a new model adapted to the situation we are currently experiencing.***

Keywords: Epidemic modeling, compartmental models, global warming, microorganisms, emerging and re-emerging infectious diseases, new dynamic process of infection, Simulation, equilibrium point, basic reproduction number, stability, Lyapunov function.

1. Introduction

Mathematical modelling of a problem is a very necessary tool for simulating experiments that are not feasible (for cost reasons) and for predicting scenarios. In the field of epidemiology, researchers such as (L.Q.Gao(1995)), (Zohra (2016)), (Bentout (2019)) are very satisfied with compartmental models, because these models are easier to handle and solve. These models require the division of the population into a number of categories according to disease status (susceptible to infection, non-contagious infected, contagious infected, immune, deceased, etc.) (Antoine (2008)). The change in the number of individuals in each of these boxes is governed by a set of differential equations (J.M.M.ONDO (2012)). To take more to the study of infectious diseases that have behavior of the infected individual, remains and remains contagious until the end of his life. (L.Q.Gao (1995)) proposed the famous SEI model. The analysis ofthe stability of this model is then proposed by (A.Korobeinikov (2004)) using the Lyapunov functions. Moreover, today's global warming is generating phenomena to modify the behaviour of our biodiversity and ecology, such as : the capacity to multiply or to pass the bad season of microorganisms (B.Marçais and al. (2000)), the epidemic development of many parasites (B.Marçais and al. (2000)), the mutation and combination of viruses (NDAFA (2017)), the resistance of micro-organisms or pathogenic agents (Muylaert A. (2012)), (Boerlin and D.G. (2006)). All these phenomena have a major impact on the large-scale spread of the disease and the inadequacy of existing epidemic propagation models in the face of the new behaviour of emerging infectious diseases, for example the SEI model.

At this stage, we are reaching the limits of our knowledge on the links between biodiversity and the ecology of transmission of infectious agents. The whole world and the international organisation are asking. This has led (S.Morand and Lajaunie (2015))(column 3, paragraph 1, page 1) in its work to announce that *"if the transmission of an infectious agent depends on local conditions of biodiversity, it is necessary to build models integrating the modifications of biodiversity with climatic variables"* and also the organisation (CCE (2009)) (paragraph 7, page 22) to leave a perspective in its document of *"Strengthening the capacities for modelling the effects of extreme meteorological phenomena on health"***.** We are interested in this issue. This paper investigates the prediction of new behaviour of mutated infectious agents by global warming and proposes the model to adapt to emerging infectious disease caused by mutated infectious agents. In the following, our work is divided into five sections. Section 2 elaborates the new dynamic process of infection. Section 3 proposes the new definition of our hypothesis. Section 4 elaborates the new model for the spread of the epidemic and the different complementary studies. Section 5 provides a conclusion.

1.1 New dynamic process of infection of emerging and re-emerging infectious diseases

We accept that an individual is affected by an infectious disease when he or she comes into contact with a pathogen, which can be of various kinds (an infected individual, a mosquito, a well, etc.). But we note that the modification and genetic change of micro-organisms or pathogens caused by global warming will lead to the advancement or acceleration of the period of contagiousness (we call **this early or premature contagion or precontagion**) (See figure (1)). That is, pathogens have the ability to adapt and spread very rapidly from one individual to another. This means that the infectious disease spreads not only through the symptomatically ill individual but also through the asymptomatic individual. This will cause the epidemic to spread very rapidly. It is considered here that the change in the mode of transfer of infection brought about by **new**

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pathogen behaviour does not change **the total duration of disease contraction** in the individual. But, it does increase the time of contagiousness and decrease the latency period.

The character **precontagious** of an individual is acquired only after a period of time of **latency** after infection. And the **infected** individual also remains **infectious** for some time or until death.

1.2 Mechanism of transmission of an infectious disease

The mechanism of transmission of an emerging infectious disease involves the following steps:

- Global warming has increased the temperature of the earth's surface.
- The increase in temperature has impacted the environment of living beings, including microorganisms.
- In the micro-organisms, those which are not killed by the rise in temperature, have managed to adapt, to mutate and they have sought the new favourable environment to live in (in the human organism).
- Once in human organisms, the mutant micro-organism is able to adapt and multiply very quickly.
- After the latent phase, without having yet to cause the prodrome in the host organism, they can already contaminate other organisms from saliva, sexual intercourse, sneezing, blood, some ordinary coughs, ...That is, the individual who seems to be in good health (who does not feel that he is infected with the disease) can infect the population if he is already infected.
- The infected individual remains contagious until the onset of symptoms of the disease and has continued to be contagious for some time or until death.

In order to provide our solution to the study of the modelling of this phenomenon, we had to establish the following definitions of assumptions that complement the definition of the susceptible, exposed, infected and latent compartments in the literature.

Definition of assumptions

Definition 1: *An individual who has been infected with the disease pathogen and can also transmit the disease, but has no symptoms, is referred to as a* ***precontagious or precontaminated*** *individual.*

Definition .2.*Precontaged* *represents a compartment where the disease requires a period of pre-contagion. Pre-contagious individuals are capable of transmitting the disease into the population, but they do not yet show symptoms of the disease. They are therefore assigned to this compartment with the rate* k *called* ***precontagiousness rate****. In the following, the letter* P *will be used to refer to individuals who are infected and contagious, but do not yet represent symptoms of the disease.*

Definition 3: *The* period of ***precontagiousness*** *is a period of time when an infected individual does not yet show symptomatic signs of the disease, but can transmit the disease to another individual.*

Definition 4: *The* ***infected*** *compartment represents those who are not only already infected and have shown symptoms of the disease, but are also capable of transmitting the disease back into the population.*

Definition 5: *The* **period of contagiousness** *is a distinct phase of time when the sick individual (person with symptoms of the disease and whose health is impaired) transmits a disease to the other individual.*

In the figure (1), we illustrate the context in which this event takes place : This schematic presentation shows the different phases (disease states).

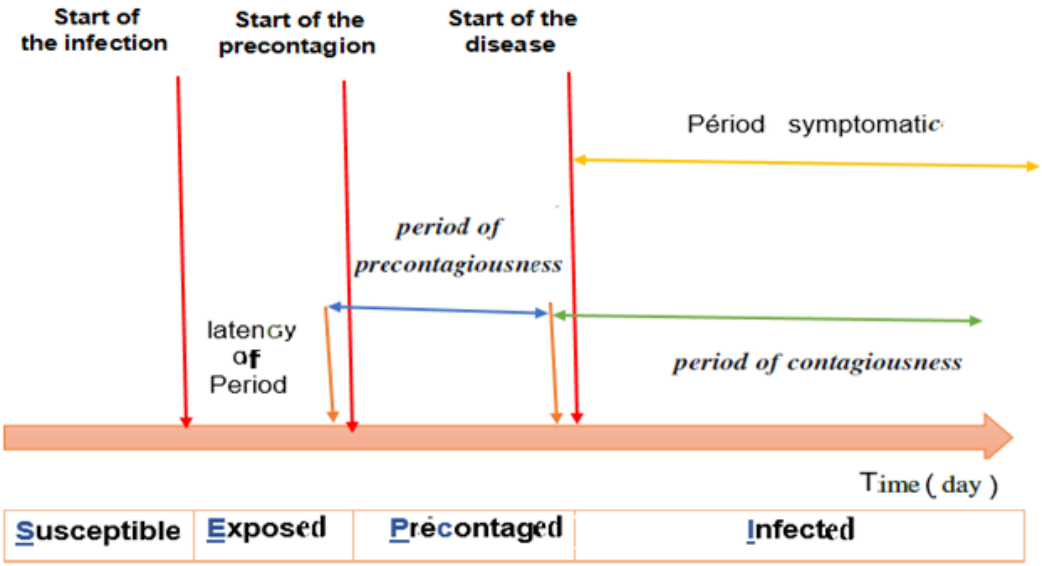

Figure 1: Representation of the contagion process

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2. Proposed models of epidemic spread

In this section, we focus on modelling the new phenomenon of the spread of infectious diseases caused by global warming in the population. We highlight the new dynamic process of infection of emerging infectious diseases on the one hand, and the different hypotheses of infectious disease behaviours on the other hand. Our work consists in proposing propagation models capable of understanding the different behaviours of the infectious disease and the new mechanism of the rapid diffusion of the infection. The model assumes that the population is constant. It is also homogeneous (no age structure, no spatial and social structure).

2.1 Objectives of propagation models

The main objective of our work is to develop new compartmental models by integrating the above new dynamic infection processes into the SEI compartmental model in the literature.

This integration effectively contributes to the modelling and simulation of any form of emerging diseases caused by global warming. We model the phenomenon of the spread of the emerging infectious disease. Other more specific objectives are envisaged by setting up the SEPI models and studying: the simulation of the model, the equilibrium points, the basic reproduction number $R_0$and the stability of the equilibrium point.

In this work, epidemic modelling only considers cases where the infection spreads directly: first, between precontagious (Precontaminated) and susceptible individuals second, between infectious (infected) and susceptible individuals (no epidemic vectors).

2.2 Le modèle dynamique simple de SEPI

In our work, the Susceptible, Exposed, Precontaged, Infected (SEPI) is the first model we develop. Indeed, we assume that the epidemic spreads in a very fast way and the infected individual remains contagious until the end of his life. This type of modelling is suitable for such short interval periods that natural mortality and emigration are balanced by birth and immigration.

2.2.1 Definition of the assumptions of the dynamic model of SEPI

In order to develop our SEPI model, we make some assumptions:

- **A1**: The size of the population is equal to N, assumed fixed ;
- **A2**: The time variable t is of discrete type, such that $t \in T$ or T is the total duration of the epidemic ;
- **A3**: The time period $\Delta t = dt$ represents hours or days or weeks ;
- **A4**: At each instant t, the population N is subdivided into four compartments : S(t) : set of Susceptible individuals, E(t) : set of Exposed individuals, P(t) : set of Precontaged individuals, I(t) : set of Infected individuals with $N = S(t) + E(t) + P(t) + I(t)$ and $S(0) = S0 > 0$, $P(0) = P0 > 0$ and/or$I(0) = I0 > 0$;
- **A5**: We assume that each susceptible individual in a period of time Δt is exposed, precontaged and then infected ;
- **A6**: The transmission of the infection is done through a direct contact between: firstly, susceptible S and one or more precontaged P with a factor β_p of proportionality (also called rate of precontagion or rate of transmission or rate of transmission of the susceptible to the exposed) ; secondly, susceptible S and one or more infected I with a factor β_i of proportionality (also called rate of infection). We admit that a βfactor is the total transmission rate or of exposure such that $\beta = \beta_p + \beta_i$;
- **A7**: An infected individual remains contagious for the rest of his or her life at the rate of λ.

In the field of epidemiology, we can schematise the SEPI model by boxes or compartments. This is the subject of the following paragraph.

2.2.2 Schematic of the SEPI model

Each compartment represents the different statuses in which individuals in a population may find themselves during the disease. We note: $\beta > 0$: the rate of exposure (or of transmission from the susceptible to the exposed), $k > 0$:the rate of precontagiousness (or of transmission from the exposed to the precontaged), $v > 0$: the rate of contagiousness (or of transmission from the precontaged to the infected). The diagram of the SEPI model is illustrated in Figure (*2*):

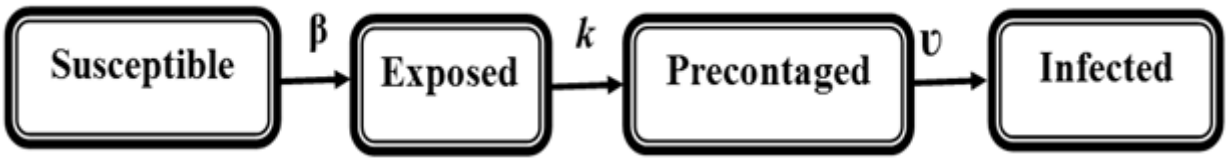

Figure 2: Scheme of the SEPI model

To these four different states, we can associate four evolutionary equations (see (1) below).

2.2.3 Representation in the form of the differential equations of the SEPI model

According to hypothesis (A7) in section (*4.2.1*) above, we consider that during the time interval *dt*, the Susceptuble compartment has lost the number S ($\beta_p P + \beta_i I$) of individuals exposed by the disease. According to the hypothesis (A6), we consider the new cases reached by the infection during the time interval *dt* which will be equal to $\beta_i S(t) I(t)$. And the new cases reached by the precontagion during the time interval *dt* which will be equal to $\beta_p S(t) P(t)$. We obtain the new cases exposed to the disease during the time interval *dt* which will be equal to $\beta_p S(t) P(t) + \beta_i S(t) I(t) = S(t)(\beta_p P(t) + \beta_i I(t))$. According to the hypothesis (A5), we consider that during the time interval *dt* the compartment precontaged by the disease has increased in number *k*E

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individuals and at the same time, it loses the number *P* of sick or infected individuals. According to the hypothesis (H5) and (A7), we consider that during the time interval *dt* the compartment Infected has increased in number *k*E of precontaged individuals. And we can present the SEPI model as a system of differential equations (1).

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta_p S(t)P(t) - \beta_i S(t)I(t) \\ \dfrac{dE(t)}{dt} = \beta_p S(t)P(t) + \beta_i S(t)I(t) - kE(t) \\ \dfrac{dP(t)}{dt} = kE(t) - \nu P(t) \\ \dfrac{dI(t)}{dt} = \nu P(t) \end{cases} \quad (1)$$

There is a unique solution for the model (1), under the initial conditions: $S(0) = S_0$; $E(0) = E_0$; $P(0) = P_0$; $I(0) = I_0$in particular, in the region, $\Omega = \{(\mathcal{S}, \mathcal{E}, \mathcal{P}, \mathcal{I}), \mathcal{S} > 0; \mathcal{P} > 0; \square > 0$which is positively invariant for the system. For by definition:

Definition 6: *A set G is said to be positively invariant if* $\forall x_0 \in G$, *the trajectory passing through* x_0*is contained in G after* x_0*: if* x *is the solution of the system* X'= F(X) *(with* F *of class* $\mathcal{C}^\infty$*) verifying* $x(0) = x_0$, *then* $\forall t \geq 0$; $x(t) \in G$.

We admit that $\frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dP(t)}{dt} + \frac{dS(t)}{dt} = 0$, we deduce from this that $\forall t \geq 0$, $S(t) + E(t) + P(t) + I(t) = S(0) + E(0) + P(0) + I(0) = N$with $N > 0$. We note X = (S; E; P; I), we can rewrite this differential system (1) in the form $X' = F(X)$ with F of class $\mathcal{C}^\infty$.

2.2.4 Simulation of the SEPI model

The different curves obtained with Scilab already give us an idea of the evolution of the epidemic. For the simulation, we consider here to have a precontaminated individual at time $t = 0$with N=10000, $\beta_p = 0; 2$, $\beta_i = 0; 1$, k=0, 4, $\nu = 0; 2$ et g=0.3. We consider a period *t* depending on the unit of transmission rates, and itis equivalent to a day or week or month. By running the simulation, we obtained the following curves :

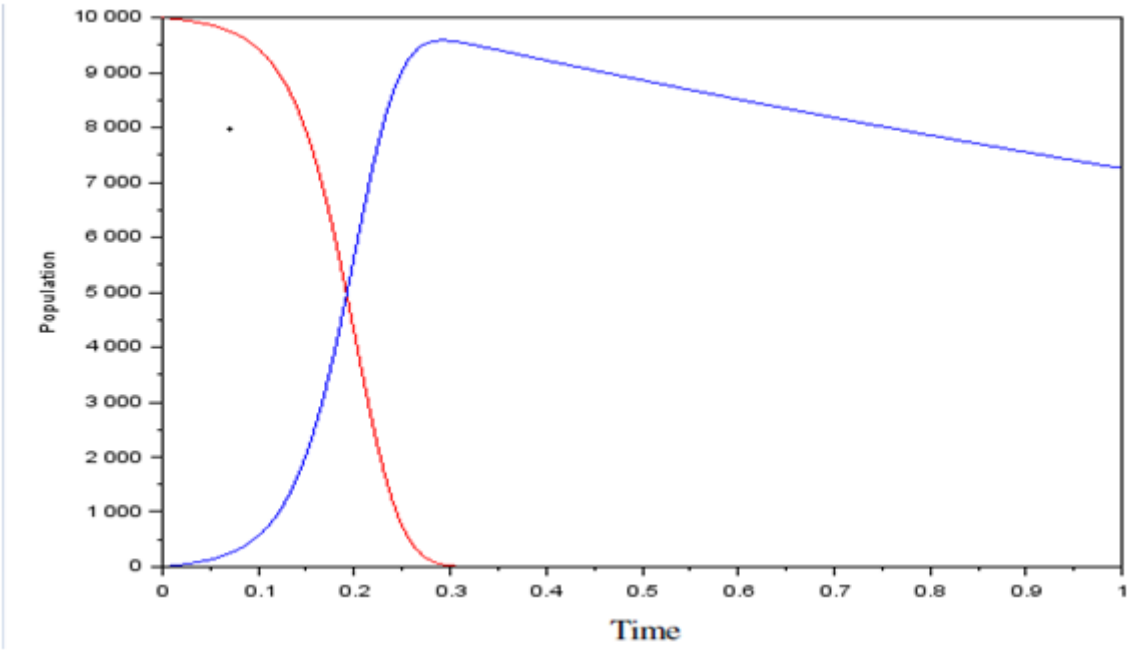

Figure 3: Curve of S(t) coloured in red and E(t) in blue

Interpretation:

From figure (3), we have shown that even with low precontagion and infection rates, the epidemic is spreading with a phenomonal and very rapid speed. The entire susceptible population is already exposed after only 0.3 of our period.

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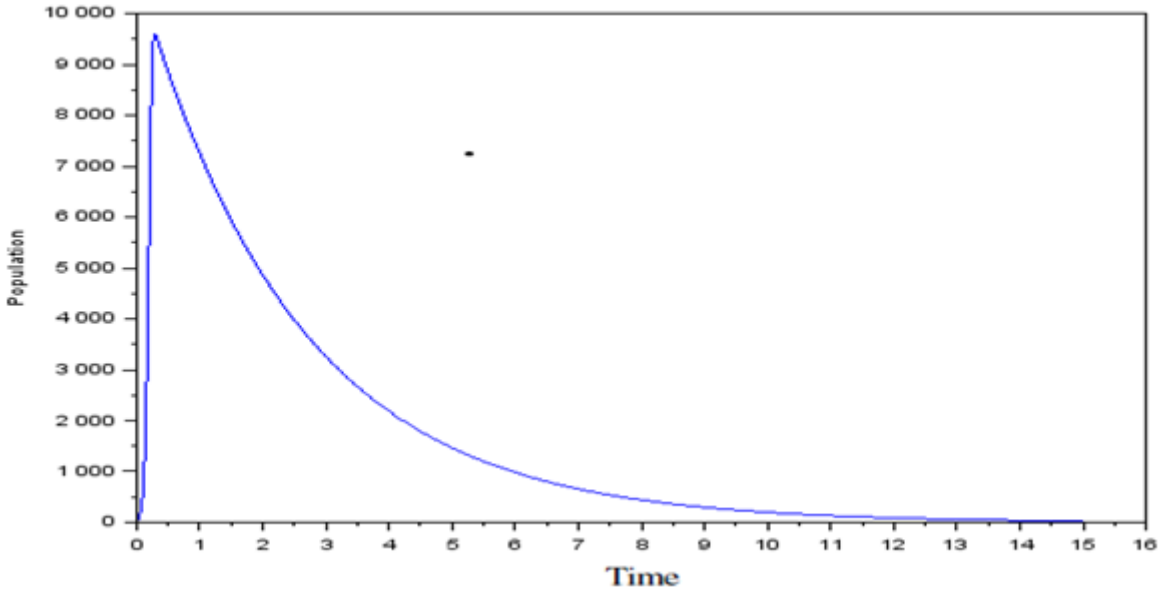

Figure 4: Curve of E(t) during a phase of the epidemic

Interpretation:
According to figure (4), after the phenomonal evolution of the epidemic, the curve of the Exposed stablilises and fades after the 15^{e}period of our epidemic.

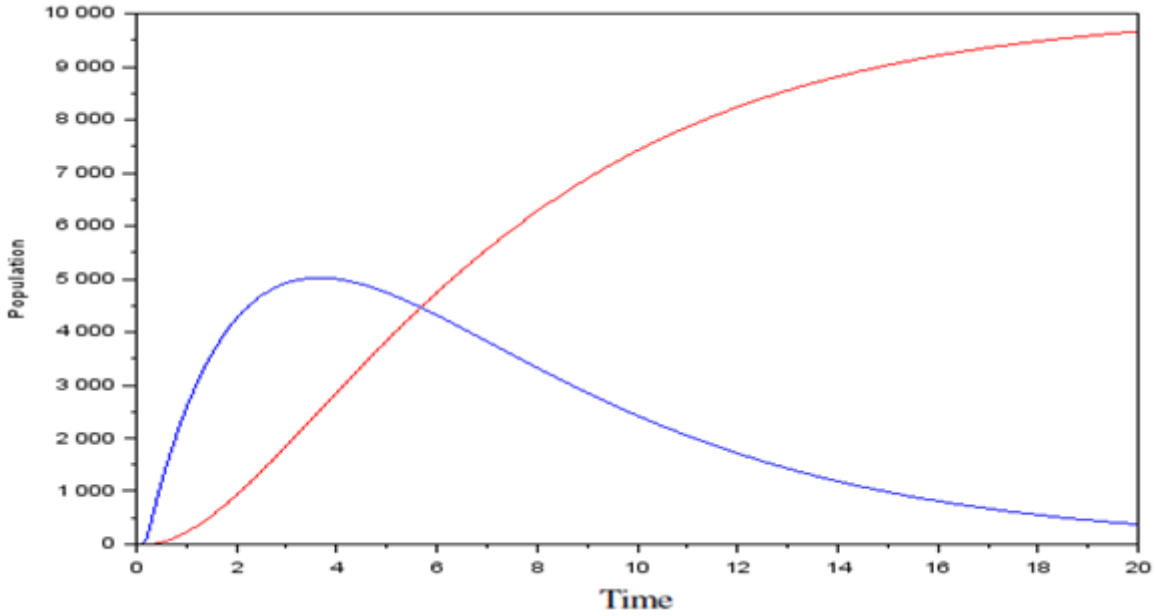

Figure 5: Curves of I(t) coloured in red and P(t) in blue

Interpretation:
From Figure (5), it appears that after the sharp increase, the curves for the Precontaged and the Infected stabilize and become endemic after the 20^{e}period.

2.2.5 Study of the equilibrium point of the SEPI model
Lyapunov in (J.M.M.ONDO (2012)), defines the equilibrium point as follows:

Definition 7: *Consider U, a non-empty open of R^n containing 0, and I a non-empty interval of* R, *not bounded on the right. Let be systems of the form:*

$$\dot{x} = f(x) \quad (2)$$
$$\dot{x} = f(t, x) \quad (3)$$

where the functions f : U $\rightarrow R^n$ *for the system* (2) and f : I × U$\rightarrow$ R^n *for the system* (3) *are assumed to be continuous.*

A point a is a point of equilibrium or state of equilibrium or singular point of the system (2) *(resp.* (3)**),** *if* f(a) = *0* (*resp. if, for any* t ∈I; f(t; a) = 0).

We then obtain the following proposition:

Proposition 1: *Let N>0. Then the system* (1)*with the condition* S(0) = S_0, E(0) = E_0, P (0) = P_0, I(0) = I_0*et S(t)+E(t)+P(t)+I(t)=S(0)+E(0)+P(0)+I(0)=N admits a unique solution (S, E, P, I) defined on*[0; +1[.

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Proof:
Equilibrium points are calculated in the absence of infection and/or precontagion. The equilibrium point of the model (1) satisfies the system below:

$$\begin{cases} -\beta_p SP - \beta_i SI = 0 \\ \beta_p SP + \beta_i SI - kE = 0 \\ kE - \nu P = 0 \end{cases} \quad (4)$$

In the absence of infection (I=0) and precontagion (P=0), we obtain the following proposition:

Proposition 2: *Let N>0, in the absence of infection (I=0) and precontagion (P=0), then the system* (1) *admits the equilibrium point :* $E_0 = (N, 0, 0, 0)^T$.

Proof:
By replacing I=0 and P=0 in the first, second and third equations in the system (4), with $S + E + P + I = N$, we obtain the first equilibrium point $E_0 = (\hat{S}, \hat{E}, \hat{P}, \hat{I})$:

$$E0 = (N, 0, 0, 0)^T \quad (5)$$

In the presence of the precontagion ($P \neq 0$) and in the absence of the infection ($I = 0$), if $\nu \neq 0$, we obtain the following proposition :

Proposition 3. *Let N>0, in the presence of the precontagion (P≠0) and in the absence of the infection (I = 0), if ν≠0, then:*

1) The system (1) admits the equilibrium point:
$$E_p^* = \left(\frac{\nu}{\beta_p}, \frac{\nu(\beta_p N - \nu)}{\beta_p(k+\nu)}, \frac{k(\beta_p N - \nu)}{\beta_p(k+\nu)}, 0 \right)^T ;$$
2) Moreover, for all t>0, we have $\beta_p N > \nu$.

Proof
Replacing $P \neq 0$and $I = 0$, if $\nu \neq 0$, the system (4) becomes :

$$\begin{cases} \beta_p SP - kE = 0 \\ kE - \nu P = 0 \quad (6) \\ \nu P \neq 0. \end{cases}$$

The second equation of (6) implies : $E^* = \frac{\nu P}{k}$and the first gives **:** $S^* = \frac{\nu}{\beta_p}$.
Since I=0, we admit that:

$$N = S + E + P \quad (7)$$

Replacing $S^*$and E^*in (7), we obtain:
$\mathbf{P^*} = \frac{\mathbf{k(\beta_p N - \nu)}}{\boldsymbol{\beta_p(k+\nu)}}$.

Replacing P^* we then have the equilibrium point $E_p^* = (S^*, E^*, P^*, 0)$ as follows:

$$E_p^* = \left(\frac{\nu}{\beta_p}, \frac{\nu(\beta_p N - \nu)}{\beta_p(k+\nu)}, \frac{k(\beta_p N - \nu)}{\beta_p(k+\nu)}, 0 \right)^T \quad (8)$$

We note that $\hat{S} > S^*$with $\hat{S}$is the susceptible of (5), implies:

$$N > \frac{\nu}{\beta_p} \quad (9)$$

From (9), we deduce that :

$$P^* = \frac{k(\beta_p N - \nu)}{\beta_p(k+\nu)} > 0 \quad (10)$$

And

$$E^* = \frac{\nu(\beta_p N - \nu)}{\beta_p(k+\nu)} > 0 \quad (11)$$

According to (9), (10) and (11) we have $\beta_p N > \nu$**.** But according to (8), if $\nu = 0$, we obtain the equilibrium point

2.2.6 The basic reproduction number R_0 of the SEPI model
From the concept of the basic reproduction number according to (G.Sallet (2010)), we have the impression that if $R_0 > 1$, then we will observe an increase in cases, thus an epidemic, and that if $R_0 < 1$ then the case will disappear. Using the condition of the study of the base number R_0in (L.Chahrazed (2002)) :

- If $R_0 < 1$, the equilibrium point E_0 is locally asymptotically stable ;
- If $R_0 < 1$, the equilibrium point E_0 is unstable.

First of all, we recall the definition of the spectral radius.

Definition 8: *The spectral radius of a matrix A is the maximum value of the modulus of the eigen values of A. We note*: $\rho(A) = \max_{\lambda \in S_p(A)} |\lambda|$, *with* $S_p(A)$ *: the set of eigen values of the matrix A.*

Definition 9: *A matrix is said to be Metzler (resp. strict Metzler) if and only if its non-diagonal terms are positive (resp. strictly positive).*

According to the work of (G.Sallet (2010)), we define the R_0as follows:

Definition 10: *(Basic reproduction rate) If the transmission matrix is stable, then we define* R_0*by* $R_0 = \rho(-FV^{-1})$*. Since V is a Metzler matrix, it is stable and implies that* $-V^{-1} \geq 0$*. This proves that-*FV^{-1}*is a positive matrix.*

To determine the R_0, one can be satisfied to consider the system on the space (*S, E, P)* since if one knows(S, E, P)**,** one knows I**,** it comes

$$\begin{cases} \frac{dS(t)}{dt} = -\beta_p S(t)P(t) - \beta_i S(t)I(t) \\ \frac{dE(t)}{dt} = \beta_p S(t)P(t) + \beta_i S(t)I(t) - kE(t) \quad (13) \\ \frac{dP(t)}{dt} = kE(t) - \nu P(t) \end{cases}$$

The biological domain is $\{(S, E, P) \mid 0 \leq S \leq N,\ 0 \leq E \leq N,\ 0 \leq P \leq N\}$. The set $\Omega = \{S, E, P \mid 0 \leq S,\ 0 \leq E,\ 0 \leq P,\ S+E+P \leq 1\}$**.** We have a variety of equilibrium points $(S,0; 0 \mid 0 \leq S \leq N)$on the S axis. Let us take an equilibrium $(S0, 0, 0,$Tor$S_0 = N)$, then we have at this point Disease Free Equilibrium (D.F.E). According to the definition (:10)**,** it is enough to consider the carriers of pathogens and (E; P) for the calculation of the jacobians, and with the notations, it comes:

$$\boldsymbol{F(E,P)} = \begin{bmatrix} \beta_p SP \\ 0 \end{bmatrix} \text{ and } \boldsymbol{\nu(E,P)} = \begin{bmatrix} -kE \\ kE - \nu P \end{bmatrix}.$$

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So $F(DFE) = \begin{bmatrix} 0 & \boldsymbol{\beta}_p \boldsymbol{S} \\ 0 & 0 \end{bmatrix}$ and $v(DFE) = \begin{bmatrix} -k & 0 \\ k & -\nu \end{bmatrix}$.

We obtain $-FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_p S}{\nu} \\ 0 & 0 \end{bmatrix}$.

So,

$$R_0 = \frac{\boldsymbol{\beta}_p \boldsymbol{S}}{\nu}. (\mathbf{14})$$

But in equilibrium or at t=0, we get

$$R_0 = \frac{\boldsymbol{\beta}_p \boldsymbol{S_0}}{\nu} = \frac{\boldsymbol{N\beta}_p}{\nu} \ (\mathbf{15})$$

2.2.7 Study of the stability of the disease-free equilibrium point or D.F.E.

According to Lyapunov's Theorem in (J.M.M.ONDO (2012)):

Consider U, a non-empty open of R^n containing 0, and I a non-empty interval of $\mathbb{R}$, not right bounded.

Definition 11: Let $t_0 \in I$ and $V: I \times U \to R$be continuously differentiable such that for all $t \in I$. Suppose that $x^* = 0$ is an equilibrium point of the system (2). If there exists a neighbourhood U_{t_0} of 0 and a function$V: U_{t_0} \to \mathrm{R},^{+}$ continuous and with continuous partial derivatives, such that:

1) V being positive definite;
2) The total derivative of V, i.e. $\dot{V}$,for the system (2), is negative, then 0 is stable for the system (2), is negative, then 0 is stable for the system;
3) If, in addition, the total derivative $\dot{V}$for the system (2) is negative, then 0 is asymptotically stable. V is, in this case, a strict Lyapunov function.

Indeed, the system (13) has a disease-free equilibrium, which is given by (S_0, 0, 0) = (N, 0, 0)**.** By studying the stability of the system, we obtain the following theorem:

Theorem 1: *If$\mathcal{R}_0 \leq 1$then the DFE is globally asymptotically stable on* Ω*.*

Proof:

Consider the Lyapunov function V(S, E, P) = E + P. We obtain:

$$\dot{\boldsymbol{V}} = \dot{\boldsymbol{E}} + \dot{\boldsymbol{P}}$$
$$= \beta_p SP - \nu P$$
$$= P(R_0 - 1)\nu$$
$$\leq 0$$

Moreover $\dot{\mathbf{V}} = 0$**,** if E+P=*0*or S=$S_0$and R_0=1. So the largest invariant set contained in this set is $\Psi = \{(S, E, P) \in \Omega \mid \dot{\mathbf{V}}\ (S, E, P) = 0$ which is reduced to the DFE. Since we are in a positively invariant compact, according to the LaSalle invariance principle in (N.P.Bhatia and G.P.Szego (1970)), the DFEis globally asymptotically stable in Ω.

2.2.8 Global stability of the endemic balance

An equilibrium for the system (13), different from the DFE, is given by (S^*, E^*, P^*) in the proposition (3),

Where

$$\mathbf{S}^* = \frac{\boldsymbol{\nu}}{\boldsymbol{\beta_p}} = \frac{\mathbf{N}}{\mathrm{R}_0}, \mathbf{E}^* = \frac{\boldsymbol{\nu}(\boldsymbol{\beta_p}\mathbf{N} - \boldsymbol{\nu})}{\boldsymbol{\beta_p}(\mathbf{k} + \boldsymbol{\nu})} = \frac{\mathbf{N}\boldsymbol{\nu}}{\mathbf{k} + \boldsymbol{\nu}}\left(1 - \frac{1}{\mathrm{R}_0}\right) \text{and} \mathbf{P}^* = \frac{\mathbf{k}(\boldsymbol{\beta_p}\mathbf{N} - \boldsymbol{\nu})}{\boldsymbol{\beta_p}(\mathbf{k} + \boldsymbol{\nu})} = \frac{\mathbf{N}\mathbf{k}}{\mathbf{k} + \boldsymbol{\nu}}\left(1 - \frac{1}{\mathrm{R}_0}\right).$$

This equilibrium is in the simplex, i.e. $0 \leq S^*$, $0 \leq E^* 0 \leq \mathrm{P}^*$and $S^* + E^* + P^* \leq N$if and only if $R_0 > 1$**.**Clearly $0 \leq E^*$, $0 \leq P^*$is equivalent to $R_0 \geq 1$**.** Now we can write $S^* + E^* + P^* = N$. This equilibrium coincide with the DFE. Then there is a unique equilibrium in the interior of the simplex if and only $\mathrm{R}_0 > 1$**.**

Theorem 2: If $R_0 > 1$, the DFE is unstable and there is a unique endemic equilibrium (S^*, E^*, P^*) which is globally asymptotically stable on the Ω domain.

Proof:

According to the concept of R_0in the section (4.2.6), if $\mathrm{R}_0 > 1$ then the DFE is unstable.

Let Ω^* be the set defined by $\Omega^* = \left\{ (\mathrm{S}, \mathrm{E}, \mathrm{P}) \mid \mathrm{S} \geq \frac{\nu}{\beta_p}, \mathrm{E} \geq 0, \mathrm{P} \geq 0, \mathrm{S} + \mathrm{E} + \mathrm{P} \leq \mathrm{N} \right.$ .The set Ω^* is a positively invariant compact. We consider on Ω^* the Lyapunov function defined by

$$\boldsymbol{V}(\boldsymbol{S}, \boldsymbol{E}, \boldsymbol{P}) = (\boldsymbol{S} - \boldsymbol{S}^*) - \frac{\boldsymbol{\nu}}{\boldsymbol{\beta}_\mathrm{p}} log \frac{\boldsymbol{\beta}_\mathrm{p} S}{\boldsymbol{\beta}_\mathrm{p} S^*} + (\boldsymbol{E} - \boldsymbol{E}^*) - \boldsymbol{E}^* log\left(\frac{\mathrm{E}}{\boldsymbol{E}^*}\right) + (\mathbf{P} - \mathbf{P}^*) - \mathbf{P}^* \log\left(\frac{P}{\mathbf{P}^*}\right).$$

It is easy to verify that V is definite positive, i.e. V *(*S, E, P *)* ≥ 0 and V $(\boldsymbol{S}^*, \boldsymbol{E}^*, \mathbf{P}^*) = 0$ if and only if *(*S, E, P *)* = $(\boldsymbol{S}^*, \boldsymbol{E}^*, \mathbf{P}^*)$.

Its derivative along the trajectories of the system (*13*) is given by:

$$\dot{\boldsymbol{V}}(\boldsymbol{S}, \boldsymbol{E}, \boldsymbol{P}) = \dot{\mathrm{S}} - \frac{\boldsymbol{\nu}}{\boldsymbol{\beta}_\mathrm{p}}\left(\frac{\boldsymbol{\beta}_\mathrm{p}\dot{\mathrm{S}}}{\boldsymbol{\beta}_\mathrm{p} S}\right) + \dot{\mathrm{E}} - \boldsymbol{E}^*\left(\frac{\dot{\mathrm{E}}}{E}\right) + \dot{\mathrm{P}} - \mathbf{P}^*\left(\frac{\dot{\mathrm{P}}}{P}\right)$$
$$= -\nu - \nu \mathrm{P} - \frac{\boldsymbol{E}^*(\beta_\mathrm{p} SP - kE)}{E} - \frac{\mathbf{P}^*(kE - \nu P)}{P}$$
$$= -\nu - \nu P - \frac{\boldsymbol{\nu}(\boldsymbol{\beta_p}\mathbf{N} - \boldsymbol{\nu})}{\boldsymbol{\beta_p}(\mathbf{k} + \boldsymbol{\nu})}\frac{(\beta_\mathrm{p} SP - kE)}{E} - \frac{\mathbf{k}(\boldsymbol{\beta_p}\mathbf{N} - \boldsymbol{\nu})}{\boldsymbol{\beta_p}(\mathbf{k} + \boldsymbol{\nu})}\frac{(kE - \nu P)}{P}$$
$$= -\nu - \nu P - \nu \mathbf{P}^*\left(\frac{\boldsymbol{\beta}_\mathrm{p} \mathrm{S}}{\mathrm{kE}} + \frac{kE}{\nu P} - 2\right)$$
$$= -\nu(1 + P + \mathbf{P}^*)\left(\frac{\boldsymbol{\beta}_\mathrm{p} \mathrm{S}}{\mathrm{kE}} + \frac{kE}{\nu P} - 2\right)$$
$$\leq 0$$
$$\leq 0$$

We conclude that $\dot{V}$is positive semi definite. The endemic equilibrium is globally asymptotically stable.

3. Conclusion

Among other things, this study has identified a new dynamic process and mechanism of infection of emerging infectious diseases. In order to have hypotheses to elaborate the

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dynamic model adapted to the new behaviour of the infectious disease caused by global warming. And modelling the spread of an epidemic brings out a threshold parameter: $R_0$and the study of an equilibrium point. In our model, it allows us to distinguish the situation and the case where the epidemic will spread and the one where it will die out. In addition, it also allows us to perform our stability study of our model.

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